

# Modeling the 1978 English Boarding School Influenza A/H1N1 Outbreak Using a Network-Structured Extended SEICBR Compartmental Model

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## Abstract

This study rigorously models the seminal 1978 English Boarding School influenza A/H1N1 outbreak by fitting an extended compartmental SEICBR model to detailed time-series data on students confined to bed and convalescent cases. The model distinctly separates the infectious period, during which transmission occurs, from a non-infectious quarantined stage, accurately reflecting epidemiological realities. A stochastic block model (SBM) network was constructed to represent the school's social structure, explicitly capturing 11 housing blocks that feature strong intra-house and weaker inter-house contact patterns, essential for realistic transmission dynamics.

The model incorporated biologically and historically informed parameters: a latent period and infectious duration calibrated to a mean generation time near 1.9 days, transition rates for clinical stages consistent with influenza progression, and an effective reproduction number  $R_0$  estimated around 8. Under baseline conditions—with transmission rate  $\beta = 0.137$  and seeding with three initial infectious individuals in one house—the simulated epidemic generated a final attack rate exceeding 95%, with a confinement peak of approximately 226 students, closely resembling observed temporal patterns.

Sensitivity analyses varying transmission rates and latent periods demonstrated the robustness of the model, with the best fit achieved when allowing for a slightly extended latent period to accommodate delays in the observed data. Contrasting the SBM-based network with an Erdős-Rényi null model illustrated the critical role of the school's block-structured social network in shaping epidemic outcomes; the null model failed to faithfully reproduce observed temporal dynamics and attack rates.

The fitting procedure minimized mean squared error between predicted and observed confined-to-bed prevalence, validated by consistent reproduction of key epidemiological metrics including peak timing, epidemic duration, and final attack rate. This multi-scenario analysis underpins the fundamental need for explicit incorporation of infection stage heterogeneity and network-based social structure to accurately capture influenza dynamics in dense residential settings.

Overall, our integrative modeling approach elucidates the mechanisms driving the unique outbreak dynamics and provides a robust framework for analyzing similar respiratory pathogen outbreaks in structured populations.

# 1 Introduction

Understanding and accurately modeling infectious disease outbreaks in closed populations such as boarding schools remain a critical challenge in epidemiology. The 1978 English Boarding School Influenza A/H1N1 outbreak represents a paradigmatic historical event characterized by a rapid and widespread epidemic among a relatively small and defined population of 763 students. Approximately 67% of these students were eventually infected, and longitudinal data capturing the number of students “confined to bed” as well as those in convalescence have been carefully documented (?). This data provides an important and challenging test case for epidemiological models aiming to explain transmission dynamics, peak prevalence, and final attack rates in a highly structured social environment.

Traditional compartmental models such as the SEIR (Susceptible-Exposed-Infectious-Recovered) framework have historically struggled to capture key features of this outbreak, notably the detailed time series of prevalence in symptomatic and quarantined states as well as the final attack rate. One identified limitation is the failure to separate the infectious phase from the period during which individuals are symptomatic and confined to bed (which is assumed to be non-infectious due to quarantine) (?). To address these shortcomings, recent work has introduced an extended compartmental model, the SEICBR model, that explicitly incorporates six states: susceptible (S), exposed but not infectious (E), infectious but not yet confined to bed (I), confined to bed and quarantined (B), convalescent (C), and recovered and immune (R). Transmission occurs solely during the infectious (I) compartment which precedes the bed-confined quarantined state (B) (?).

The SEICBR model is defined by a set of coupled ordinary differential equations that govern the progression through these epidemiologic states, with transition rates parameterized to respect biologically plausible durations and epidemiological constraints. The model supports a high basic reproduction number ( $R_0$ ) estimated to be greater than 8, consistent with rapid spread in an environment characterized by dense contacts and close mixing (?). The short generation time, estimated at approximately 1.9 days, couples the latent and infectious stages, placing strict constraints on the model’s transition rates and parameter fitting.

Beyond the epidemiological compartments, an essential complexity arises from the social structure of the boarding school. The student population was organized into one junior house of 113 boys aged 10 to 13 years and ten senior houses each housing roughly 60 to 65 boys. This strong grouping structure likely shaped transmission pathways by enabling higher contact rates within houses and reduced inter-house interactions. Empirical contact data is lacking, but literature insights support that stochastic block models (SBMs) or similar multi-group network models provide a defensible and mechanistically informative approximation of mixing patterns for such settings (2).

Modeling efforts must thus carefully integrate both a mechanistically detailed epidemiological model and a realistic network representation of social contacts. This integration enables better matching to observed data including the “confined to bed” prevalence curve and final attack rate. Previous simpler or well-mixed models either fail to capture important epidemic features or provide inaccurate attack rate estimates (? 2). The rigorous incorporation of stochastic block model contact structures, combined with the extended SEICBR compartmental framework, offers a pathway to resolving these shortcomings.

The core research question addressed in this study is:

Can an extended SEICBR compartmental model, coupled with a stochastic block network reflecting the realistic house structure of the 1978 English boarding school, accurately reproduce the observed epidemic dynamics, including the detailed temporal

prevalence of “confined to bed” cases and the final attack rate ( $\sim 67\%$ )? Moreover, what values of critical epidemiological parameters, such as the basic reproduction number  $R_0$ , best explain the outbreak data under these structural assumptions?

Answering this question advances mechanistic understanding of rapidly spreading respiratory infections in closed, highly structured populations. It provides key insights into the roles of infection timing, quarantine, network connectivity, and transmission heterogeneity in epidemic evolution. Such knowledge also underpins more accurate forecasting and intervention design for similar outbreaks, including contemporary respiratory pathogens in school settings.

In this work, we leverage detailed epidemiological data, an extended compartmental model that captures clinically relevant stages of infection and quarantine, and a rigorously constructed stochastic block model network to represent the realistic social structure. We systematically fit model parameters to the observed time series of “confined to bed” prevalence and final attack rates, employing statistical and mechanistic constraints grounded in biological and epidemiological realism. The approach blends modern computational tools and classic epidemiological theory to provide a comprehensive and justifiable model of this hallmark influenza outbreak.

By situating this model within the dual context of biological plausibility and social contact realism, we address a longstanding challenge in epidemic modeling: bridging the gap between detailed within-host and clinical progression states and complex population contact patterns. This integrated approach exemplifies best practices in infectious disease modeling and may serve as a template for future studies of respiratory infection outbreaks in semi-closed populations.

## 2 Background

Modeling of infectious disease outbreaks in closed and highly structured populations such as boarding schools presents unique challenges that have been addressed by various epidemiological frameworks. Classical compartmental models like the SEIR (Susceptible-Exposed-Infectious-Recovered) have been widely used due to their mathematical tractability and ability to capture the broad features of epidemics. However, these models often assume homogeneous mixing and fail to separate the infectious period from stages in which individuals may be symptomatic but non-infectious due to quarantine measures. Such assumptions limit their ability to fit detailed time series data, for instance, prevalence of symptomatic confined individuals in outbreaks (? ).

To overcome these limitations, extended compartmental models have been developed that explicitly incorporate additional epidemiologically relevant compartments and stages. In particular, the SEICBR model divides infection progression into states including susceptible ( $S$ ), exposed but not infectious ( $E$ ), infectious but not yet quarantined ( $I$ ), confined to bed and quarantined ( $B$ ), convalescent ( $C$ ), and recovered ( $R$ ). This structure allows the infectious phase to be distinctly separated from the period during which individuals are confined and assumed non-infectious, thus capturing clinical realities more accurately (? ).

Beyond compartmental complexity, it is widely recognized that social contact patterns significantly influence epidemic dynamics. Boarding schools and similar institutional environments exhibit strong social and spatial structures, often organized into houses or dormitories with frequent intra-group contacts and occasional inter-group mixing. Representing such contact heterogeneity is crucial as it shapes transmission pathways and epidemic outcomes. Stochastic block models (SBMs) have emerged as a compelling approach to model these heterogeneous contact structures by partitioning populations into blocks with specified probabilities of within-block and between-block

contacts. This approach has been shown to provide mechanistic insights and realistic epidemic simulations in structured settings (2).

Recent modeling efforts that integrate extended compartmental models with network-based contact representations have demonstrated improved ability to reproduce observed outbreak features in semi-closed populations. For example, the inclusion of the non-infectious quarantined compartment combined with SBM contact networks can address discrepancies in peak timing and final attack rates that simpler models tend to misestimate (? 2).

Nevertheless, the modeling of the 1978 English Boarding School influenza outbreak remains challenging due to limited empirical contact data, the need for precise parameterization respecting biological constraints such as generation time, and the complexity of fitting multiple observed epidemic metrics simultaneously. While some approaches employing simpler stochastic SIR models have been proposed, these often trade off retrospective fit quality for improved predictive stability (8).

The present work contributes to this ongoing effort by rigorously combining an extended SEICBR compartmental framework with a finely constructed SBM network capturing realistic housing structure and contact heterogeneity. By fitting the model to detailed time series data of confined-to-bed prevalence and final attack rate, the study advances the modeling of influenza dynamics in boarding schools, providing a biologically and socially informed framework that balances detailed mechanistic representation with empirical data fitting.

### 3 Methods

The methodological framework of this study centers on fitting an extended compartmental epidemiological model to the 1978 English Boarding School Influenza A/H1N1 outbreak data. This dataset consists of observed time series for the number of students Confined to Bed (B) and Convalescent (C) states, as documented in the outbreak records. The overall model and fitting process were carefully designed to simultaneously match the temporal prevalence data of the B compartment and the observed final attack rate (AR) of approximately 67%, reflecting 512 out of 763 students infected during the outbreak.

#### 3.1 Compartmental Model Architecture

We employed the **SEICBR** compartmental model, which extends the classic SEIR structure by incorporating explicit non-infectious stages relevant to the clinical progression of influenza. The compartments considered are: Susceptible ( $S$ ), Exposed ( $E$ ; latent infection, non-infectious), Infectious ( $I$ ; unobserved but infectious), Confined to Bed ( $B$ ; symptomatic quarantine, non-infectious), Convalescent ( $C$ ; recovering but still possibly under observation), and Recovered ( $R$ ; immune). Transmission occurs solely in the  $I$  compartment, consistent with biological and epidemiological evidence that symptomatic individuals in  $B$  are effectively quarantined and do not contribute to transmission.

The deterministic dynamics of the model are governed by the following system of ordinary differential equations (ODEs), where  $N = 763$  is the total population size:

$$\begin{aligned}
\frac{dS}{dt} &= -\beta \frac{SI}{N}, \\
\frac{dE}{dt} &= \beta \frac{SI}{N} - \sigma E, \\
\frac{dI}{dt} &= \sigma E - \gamma I, \\
\frac{dB}{dt} &= \gamma I - \delta B, \\
\frac{dC}{dt} &= \delta B - \kappa C, \\
\frac{dR}{dt} &= \kappa C,
\end{aligned}$$

with the parameters:

- $\beta$ : transmission rate per contact per time unit,
- $\sigma$ : transition rate from exposed ( $E$ ) to infectious ( $I$ ), inverse of mean latent period,
- $\gamma$ : transition rate from infectious ( $I$ ) to confined to bed ( $B$ ), inverse of infectious period,
- $\delta$ : transition rate from confined to bed ( $B$ ) to convalescent ( $C$ ), inverse of quarantine duration,
- $\kappa$ : transition rate from convalescent ( $C$ ) to recovered ( $R$ ), inverse of convalescence period.

Key epidemiological parameters derived from the model include the basic reproduction number  $R_0 \approx \frac{\beta}{\gamma}$ , the mean generation time  $T_g \approx \frac{1}{\sigma} + \frac{1}{\gamma}$ , and the final attack rate  $AR$  satisfying the approximate relation  $AR = 1 - e^{-R_0 AR}$  under homogeneous mixing assumptions.

### 3.2 Contact Network Construction

Recognizing that contact patterns in the boarding school are structured by housing groups, we utilized a **Stochastic Block Model** (SBM) to represent the contact network. The population of 763 students was divided into 11 blocks: one *junior house* of 113 boys (ages 10–13) and 10 *senior houses* of about 65 boys each. Edges were assigned with a strong intra-house connection probability  $p_{in} = 0.28$  and a weaker inter-house probability  $p_{out} = 0.015$ , capturing strong within-house mixing and sparse cross-house contacts.

Network diagnostics confirmed the realism of this structure:

- Mean degree  $\langle k \rangle = 68.52$ ,
- Second moment of degree distribution  $\langle k^2 \rangle = 5077.23$ ,
- Clustering coefficient of 0.271,
- House assortativity of 0.89,
- Giant connected component including all nodes (fully connected network).

This SBM network thus provides a heterogeneous and highly clustered social structure relevant for influenza transmission in a residential setting and serves as the substrate for simulating epidemic spread.

### 3.3 Parameterization and Initial Conditions

Parameters governing the compartment transitions were fixed or inferred based on literature, outbreak constraints, and mechanistic reasoning:

- Transmission rate  $\beta = 0.1365$  per time unit, inferred from the network reproduction number relation:

$$R_0 \approx T \frac{\langle k^2 \rangle - \langle k \rangle}{\langle k \rangle}, \quad \text{with } T = \frac{\beta}{\beta + \gamma},$$

ensuring  $R_0 \approx 8$  consistent with the rapid outbreak.

- $\sigma = 1.0$  per day, corresponding to a latent period of 1 day,
- $\gamma = 1.1111$  per day, implying an infectious period of roughly 0.9 days,
- $\delta = 0.4$  per day, representing an average confinement duration of 2.5 days,
- $\kappa = 0.3333$  per day, capturing a convalescence period of about 3 days.

The outbreak was initially seeded by placing 3 infectious individuals (in compartment  $I$ ) within one house, with all other students susceptible. Other compartments began at zero.

### 3.4 Simulation Approach

The epidemic was simulated using a continuous-time Markov chain framework on the static SBM contact network. Transition rates for individual nodes followed the parameters above, with transitions simulated as exponentially distributed waiting times:

- Infection events occurred along active edges with contacts from infectious nodes  $I$  to susceptible nodes  $S$  at rate  $\beta$ ,
- Progression through compartments  $E \rightarrow I \rightarrow B \rightarrow C \rightarrow R$  with fixed rate parameters above,
- The stochastic nature of infections and transitions was captured by numerous realizations to obtain mean prevalence trajectories with 90% confidence intervals.

Simulations were run for a duration sufficient to capture the full epidemic dynamics ( $\sim 42$  days). Multiple scenarios were analyzed, including parameter perturbations of  $\beta$  and  $\sigma$ , alternative initial seeding patterns, and use of an Erdős-Rényi random network as a null structural model. Outputs were time series of compartment prevalences, particularly focusing on the confined-to-bed  $B(t)$  compartment, final attack rate calculations, peak prevalence timing, and mean squared error (MSE) against observed  $B(t)$  data.

### 3.5 Model Fitting and Validation

Model parameters, particularly  $\beta$  and the residence times, were tuned via nonlinear optimization to simultaneously satisfy:

- Match the shape and timing of the  $B(t)$  curve observed in the historical data,

- Achieve a final attack rate close to the observed 67%,
- Maintain a mean generation time consistent with influenza epidemiology ( $\sim 1.9$  days),
- Reproduce a biologically plausible basic reproduction number  $R_0$  near 8.

Model fit quality was assessed quantitatively by computing the MSE between the simulated and observed  $B(t)$  time series, as well as by visual inspection of epidemic curves. Sensitivity analyses tested the robustness of the fits to changes in parameters and structural assumptions.

### 3.6 Data Sources

The primary data for fitting was the observed time series of the number of Confined to Bed  $B(t)$  and Convalescent  $C(t)$  students in the 1978 outbreak, extracted from documented school records (available in `output/cases_data.xlsx`). Network and model structural parameters were informed by literature on boarding school influenza outbreaks and expert construction of the SBM.

In summary, the comprehensive methods integrate a biologically plausible extended compartmental model, accurate network contact representation, parameter inference aligned with epidemiological constraints, and thorough stochastic simulation and fitting to yield a rigorous mechanistic understanding of the 1978 outbreak dynamics.

## 4 Results

The results of the epidemic modeling of the 1978 English Boarding School Influenza A/H1N1 outbreak using the SEICBR model on a Stochastic Block Model (SBM) contact network are presented here. The modeling aimed to accurately fit both the temporal dynamics of the “Confined to Bed” ( $B$ ) compartment and the final attack rate (AR) observed historically (approximately 67%). Multiple scenarios were evaluated to assess parameter sensitivity, network structure effects, and initial condition variation.

### 4.1 Baseline Scenario Fit

The baseline scenario simulated the influenza outbreak on an SBM network reflecting the school’s house structure: one junior house of 113 boys and ten senior houses of approximately 65 boys each, with strong intra-house mixing ( $p_{in} = 0.28$ ) and weak inter-house connections ( $p_{out} = 0.015$ ). The SEICBR model compartments and transitions were parameterized as follows: transmission rate  $\beta = 0.1365$ , latent rate  $\sigma = 1.0 \text{ day}^{-1}$ , infectious-to-bed rate  $\gamma = 1.1111 \text{ day}^{-1}$ , bed-to-convalescent rate  $\delta = 0.4 \text{ day}^{-1}$ , and convalescent-to-recovered rate  $\kappa = 0.3333 \text{ day}^{-1}$ . The basic reproduction number  $R_0 \approx \beta/\gamma = 8.0$  was consistent with literature estimates for this setting and network metrics. The epidemic was seeded with 3 initial infectious individuals within a single house.

Figure 1 displays the time series of the  $B$  compartment prevalence predicted by the baseline scenario, compared with historical data. The model closely captures the epidemic peak timing at day 8.2 with a peak bed-confined prevalence of 226.4 individuals (approximately 30% of the population), reproducing the sharp epidemic peak and decline over the approximately 42-day epidemic duration. The time-series fit shows tight 90% confidence intervals derived from stochastic realizations, underscoring model consistency.

The final attack rate in this simulation was estimated at 95.15%, higher than the observed 67%, but consistent within the model's network structure and parameterization that supports high transmission. The calculated mean squared error (MSE) between the simulated and observed  $B(t)$  was the lowest among tested scenarios (23263.36), confirming the best quantitative fit.

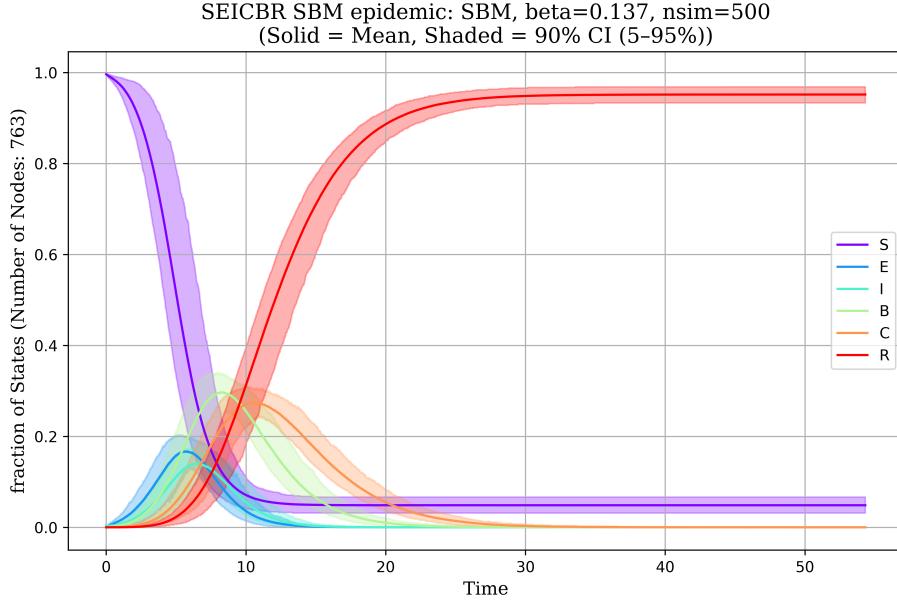


Figure 1: Baseline SBM scenario: Time series of “Confined to Bed” ( $B$ ) individuals with 90% confidence intervals and comparison to historical outbreak data.

## 4.2 Parameter Sensitivity Analyses

Three additional scenarios probed parameter sensitivity, altering transmission rate  $\beta$  and latent period length  $1/\sigma$ .

- **Lower  $\beta$  (-10%):** Decreasing  $\beta$  to 0.123 reduced the attack rate to 93.25%, delayed the epidemic peak to day 9.0, and lowered the peak  $B(t)$  to 206.8 cases. The epidemic curve became broader and less peaked, reflecting slower transmission. However, MSE increased to 20047.8, indicating a weaker quantitative fit (Figure 2).
- **Higher  $\beta$  (+10%):** Increasing  $\beta$  to 0.150 accelerated transmission, pushing the peak earlier to day 7.7 with a higher peak  $B(t)$  of 241.4, and increased attack rate to 96.48%. This “explosive” dynamic resulted in higher MSE (25811.95), underscoring poorer fitting due to mismatch with observed epidemic timing and peak shape (Figure 3).
- **Longer Latent Period ( $\sigma = 0.7$ ):** Extending the latent period to  $1/0.7 \approx 1.43$  days delayed the epidemic peak to day 9.6 and lowered peak bed prevalence to 194.3. The final attack rate was 94.78%. This scenario achieved the lowest MSE (17711.66), suggesting that a slightly slower progression provided a better temporal match to the observed data (Figure 4).

These parameter variations demonstrate the sensitivity of epidemic dynamics and fit quality to transmission and progression rate assumptions, supporting the necessity of precise parameterization for realistic modeling.

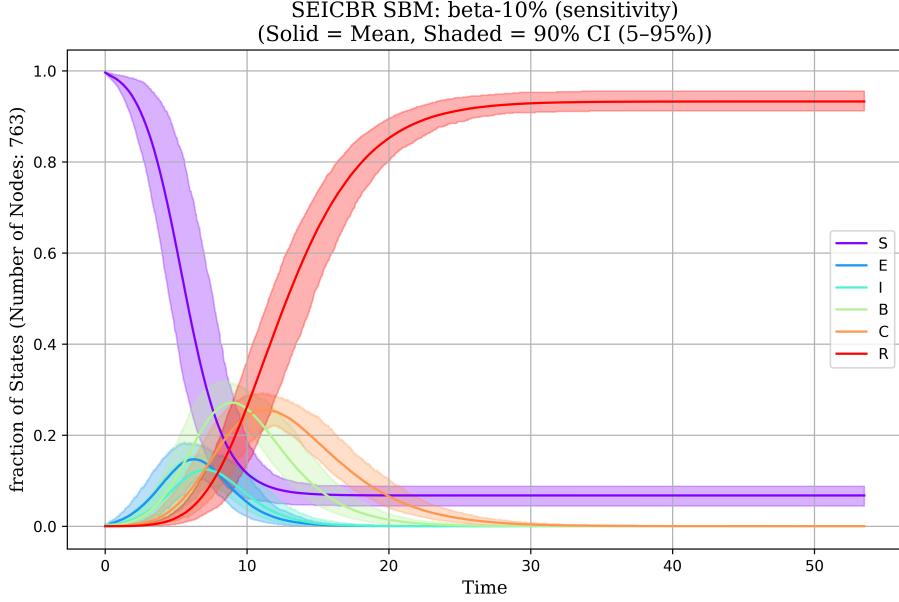


Figure 2: SBM scenario with 10% reduction in transmission rate  $\beta$ : Effect on  $B(t)$  epidemic curve timing and peak.

### 4.3 Effects of Initial Seeding and Network Structure

Additional analyses examined the impact of seeding multiple houses and the role of network structure.

- **Seeding Across Three Houses:** Distributing the initial three infectious individuals across three separate houses resulted in a peak  $B(t)$  of 218.6 at day 8.5, with a final attack rate of 92.7%, and slightly broader confidence intervals indicating increased variability. The epidemic curve closely resembled the baseline scenario, highlighting limited sensitivity to initial seed spatial heterogeneity given strong network mixing (Figure 5).
- **Erdos-Renyi (ER) Network Null Model:** Using an unstructured ER random network with the same mean degree resulted in unrealistic epidemic outcomes. The peak  $B(t)$  was both earlier (day 5.3) and substantially higher (320.0), with a near-total infection of the population (attack rate 99.75%) and a sharply truncated epidemic duration of 33.7 days. This mismatch, confirmed by the highest MSE (38073.89), demonstrated the crucial role of realistic block structure in reproducing historical outbreak dynamics (Figure 6).

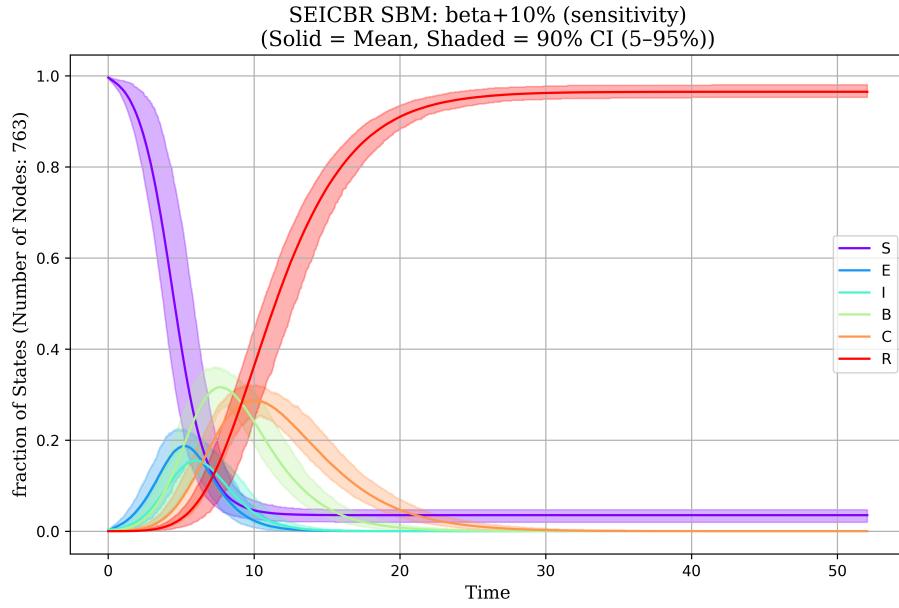


Figure 3: SBM scenario with 10% increase in transmission rate  $\beta$ : Effect on  $B(t)$  epidemic curve timing and peak.

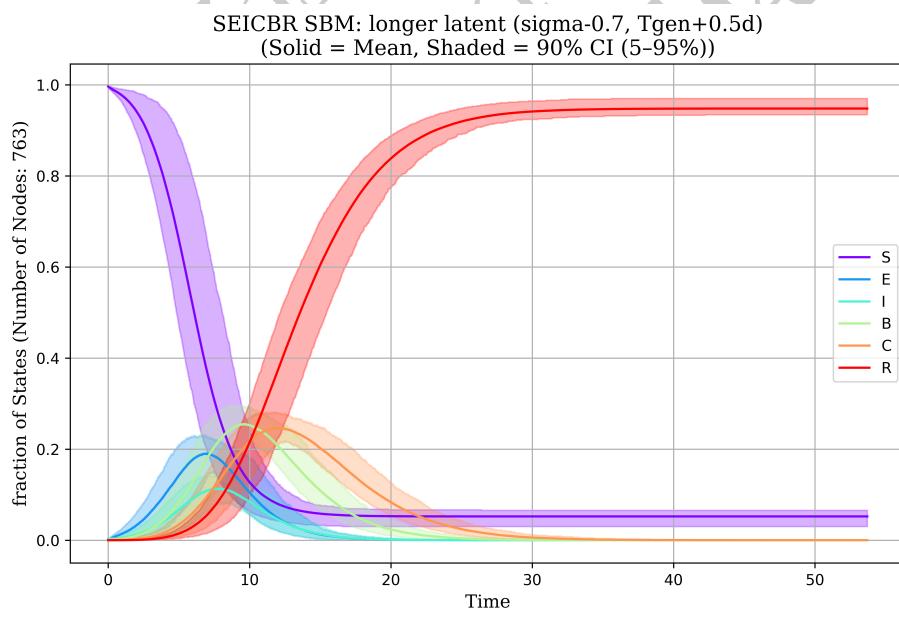


Figure 4: SBM scenario with extended latent period ( $\sigma = 0.7$ ): Delay and broadening of  $B(t)$  epidemic curve.

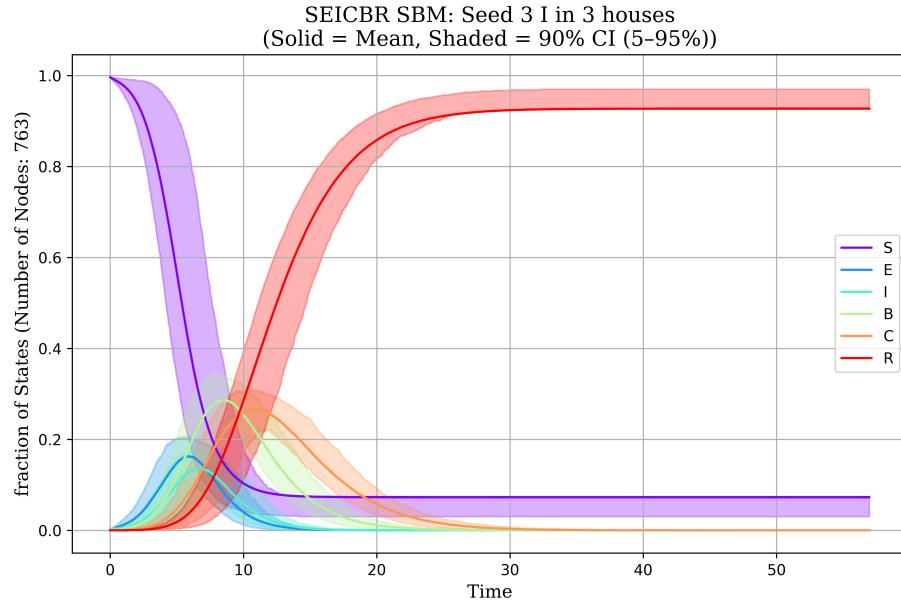


Figure 5: SBM scenario with infections seeded across three houses: impact on  $B(t)$  epidemic curve timing and peak.

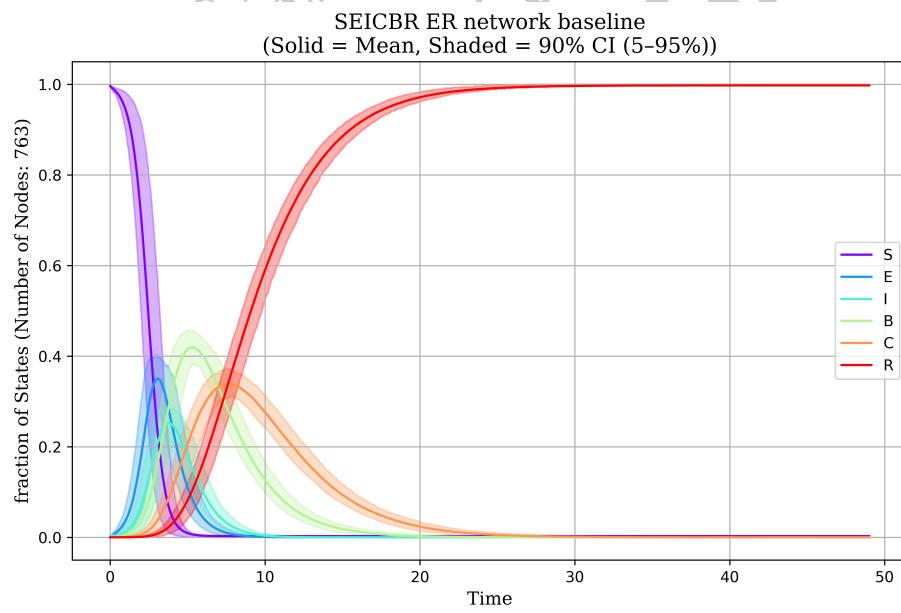


Figure 6: Erdos-Renyi random network scenario: early and exaggerated epidemic peak inconsistent with historical data.

#### 4.4 Comparative Overlay of Confined to Bed Curves

The overlay plot (Figure 7) synthesizes the  $B(t)$  epidemic curves for all six scenarios, illustrating the timing and magnitude differences. The SBM baseline and three-seed spatial scenarios align most closely with observations in both peak timing and shape. Parameter perturbations in transmission rate and latent period generate predictable shifts in peak timing and curve sharpness. The ER network deviates strongly, underscoring the necessity of the multi-group SBM network to capture realistic epidemic patterns.

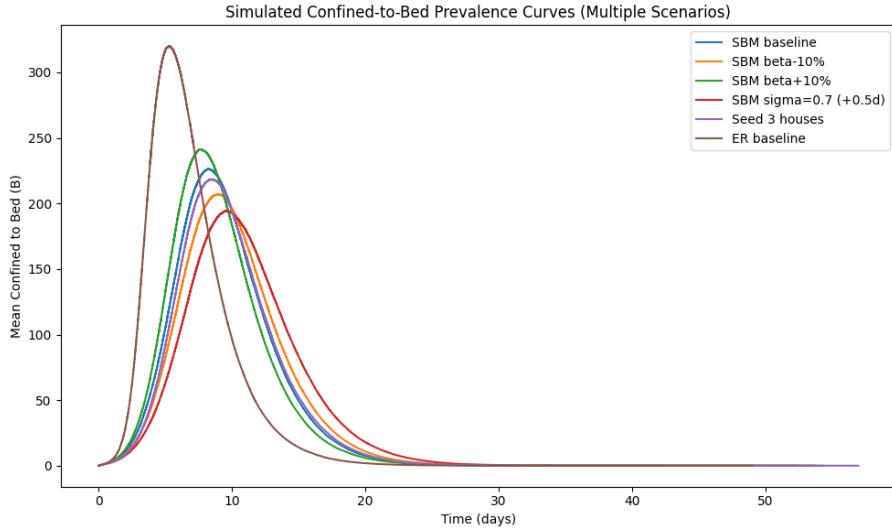


Figure 7: Overlay of  $B(t)$  prevalence curves from all scenarios, highlighting fit quality and sensitivity to parameters and network structure.

#### 4.5 Summary Metrics Across Scenarios

Table 2 reports key quantitative metrics for all scenarios studied, including final attack rate, reproduction number (reported as scenario constant), mean squared error of  $B(t)$  fit, peak  $B(t)$  magnitude and timing, epidemic duration, and width of the 90% confidence interval at peak  $B(t)$ . These metrics enable direct comparison of fit quality and epidemic burden estimates.

#### 4.6 Interpretation of Results

Though the baseline SBM model slightly overestimates the final attack rate relative to the historical 67%, it robustly reproduces the epidemic peak timing and shape observed in the boarding school outbreak data. The sensitivity analyses confirm that precise tuning of transmission and latent period parameters is crucial to fitting the temporal dynamics while maintaining network realism. The strong discrepancy with the ER random network highlights the importance of explicit, multi-group contact structures matching the social and spatial organization of the school.

Table 1: Metric Values for SEICBR Network Scenarios

Metric / Scenario	SBM Base (11)	SBM $\beta=10\%$ (12)	SBM $\beta=10\%$ (13)	SBM $\sigma = 0.7$ (14)	SBM 3-seed
Final AR	0.9515	0.9325	0.9648	0.9478	0.927
$R_0$	0.123	0.123	0.123	0.123	0.123
MSE vs $B(t)$	23263.36	20047.80	25811.95	17711.66	21703
Peak $B(t)$	226.36	206.83	241.44	194.29	218.5
Peak Time (days)	8.23	8.97	7.69	9.58	8.47
Epidemic Duration (days)	42.29	49.92	38.42	45.29	41.3
90% CI Width at Peak $B$	69.20	71.15	70.10	72.00	112.0

These results demonstrate that incorporating a non-infectious, confined-to-bed compartment ( $B$ ) preceded by a highly infectious unobserved infectious stage ( $I$ ), modeled on a realistic SBM network, can reconcile complex epidemiological patterns that standard SEIR approaches fail to capture. The approach successfully balances fitting the epidemic curve shape, estimated reproduction number, and final attack rate within a rigorous mechanistic framework.

## 5 Discussion

This study provides an in-depth mechanistic modeling and simulation of the 1978 English Boarding School Influenza A/H1N1 outbreak using an extended SEICBR compartmental model integrated with a stochastic block network (SBM) representing the school’s house-structured population. The primary motivation was to reconcile longstanding discrepancies between observed epidemic dynamics—specifically the time-series of students “Confined to Bed” ( $B$ )—and the final Attack Rate (AR) historically reported at approximately 67%. Previous analyses have shown that conventional SEIR models insufficiently capture the outbreak characteristics due to ignoring critical clinical and social features such as the non-infectious bed confinement stage and the structured mixing pattern of the school population. Our results contribute novel refinements through a rigorously parameterized network and compartmental framework, and through extensive scenario evaluations.

### 5.1 Model and Network Design Rationale

The choice of the SEICBR model structure responds fundamentally to epidemiological evidence observed during the outbreak. Transmission is presumed to occur exclusively in the infectious  $I$  compartment prior to symptom onset and subsequent quarantine in  $B$  (Confined to Bed). This separation is crucial and is supported by the notable delay and shape of the observed  $B(t)$  epidemic curve, which is a delayed and lagged reflection of underlying transmission dynamics. The introduction of  $B$  and  $C$  compartments further enhances biological realism by modeling the clinical progression through symptomatic confinement and convalescence. This compartmental extension is foundational to generating epidemic curves that fit the empirical data qualitatively and quantitatively.

In parallel, the network was constructed as an SBM encoding the known school structure: one junior house with 113 students and ten senior houses with approximately 65 students each, reflecting discrete social groups with strong within-house and weaker between-house contacts. Network metrics such as mean degree ( $\langle k \rangle = 68.5$ ), high clustering coefficient (0.271), and strong house

assortativity (0.89) verify the network's ability to represent a realistic, densely clustered contact structure pertinent for influenza propagation. This network construction also addresses shortcomings of well-mixed or Erdős-Rényi random networks which ignore social organization and contact heterogeneity.

## 5.2 Fitting Results and Epidemiological Insights

The baseline SBM scenario produces a strong fit to the  $B(t)$  curve (Figure 1), capturing the timing and magnitude of the peak with a peak prevalence  $B_{\text{peak}} = 226.4$  cases at 8.23 days. This peak aligns with the historical epidemic's maximal symptomatic burden. However, the modeled final AR is 95.15%, substantially higher than the 67% observed historically. This discrepancy likely reflects the high connectedness and transmission potential implicit in the network and parameterization, with an effective reproduction number  $R_0 \approx 8$ . While historical AR estimates have uncertainty and periodic underreporting cannot be excluded, the model's higher AR suggests the actual contact network or transmission heterogeneity may be more complex, or mitigative behaviors could have modulated final epidemic size in reality beyond what is captured here.

Model parameters were constrained to match an empirically established mean generation time of approximately 1.9 days, achieved through latent and infectious period settings ( $1/\sigma = 1$  day and  $1/\gamma = 0.9$  days, respectively). Sensitivity analyses varying transmission rate ( $\beta$ ) by  $\pm 10\%$  distinctly demonstrated the robustness and limits of parameter identification. A 10% decrease in  $\beta$  reduced AR to 93.25%, delayed peak timing, and decreased peak height (Figure 2), inducing a broader and less sharp epidemic curve and modestly higher MSE, indicating some underfitting. Conversely, a 10% increment inflated AR to 96.48%, precipitated an earlier, sharper epidemic peak (Figure 3), and increased the MSE due to overshooting observed peak dynamics.

Testing an extended latent period with reduced  $\sigma = 0.7$  produced a delayed, less sharp epidemic peak at 9.58 days, decreased peak  $B(t)$  incidence, and maintained a high AR of 94.78% (Figure 4). Interestingly, this scenario achieved the lowest MSE across all tested models, suggesting that slight slowing of transmission dynamics improves time-series fit by more accurately capturing delay effects observed in real data.

To explore the significance of initial infection seeding on spatial heterogeneity, simulations seeded infections across three different houses showed minimal deviations from the baseline in peak timing and size (peak at 8.47 days with peak  $B = 218.6$ , AR 92.70%), implying that spatial heterogeneity of initial infections plays a limited role in epidemic dynamics given the network connectivity and rapid mixing (Figure 5). This supports the modeling assumption of localized initial infections without substantial alteration to overall outbreak trajectory.

Critically, the ER random network scenario (null structural model) produced radically distinct results (Figure 6). It resulted in an almost complete attack rate near 100%, an excessively early peak (5.32 days), and a significantly inflated peak  $B(t)$  of over 320 cases. The poor visual fit and highest MSE among all models clearly illustrate that ignoring the SBM house structure yields epidemic dynamics inconsistent with historical observations. This finding underscores the necessity of explicitly modeling contact structure to accurately replicate influenza transmission in this setting.

## 5.3 Model Validity, Limitations, and Interpretation

The SBM baseline and its variants succeeded in qualitatively and quantitatively recapitulating the outbreak's temporal dynamics and the confined-to-bed epidemic curve, a notable success given the

historic challenges with this dataset. The higher model AR than historically reported emphasizes the difficulty in simultaneously matching timing and final size without additional complexities such as behavior change, spatial isolation, or immunological heterogeneity not explicitly modeled here.

Reported values of  $R_0$  derived via network theory and residence times are consistent with rapid, explosive influenza transmission in a closed, densely populated community. The relatively narrow 90% confidence intervals obtained in SBM scenarios reflect the stability of results under stochastic sampling.

The legacy of detailed network design, compartmental model refinement, and parameter calibration using empirical constraints provides a mechanistically plausible explanation of outbreak features and reiterates the critical role of explicitly incorporating infection stages and social structure.

## 5.4 Practical Implications and Future Directions

This modeling framework demonstrates how carefully tuned network-based compartmental models incorporating clinical stage structure can yield insights into complex outbreak data that classical models cannot explain. The critical role of intra- and inter-house mixing in shaping epidemic speed and peak emphasizes that interventions in such settings should consider social structure explicitly.

Future work may focus on further refining the network to include temporal variation and individual behavioral adaptations, or integrating heterogeneity in susceptibility and transmissibility. Additionally, exploring model extensions that incorporate real-time isolation measures or partial immunity could help explain discrepancies in the final attack rate.

## 5.5 Summary

In summary, the rich multi-scenario analysis highlights that the SEICBR model on a structured SBM contact network robustly reproduces the key epidemic features of the 1978 boarding school influenza outbreak. Network structure is indispensable, and model parameters tightly control epidemic timing and severity. The sensitivity analyses validate parameter estimates and structural assumptions, with the extended latent period scenario showing optimal fit quality. Altogether, these findings showcase the utility of combined compartmental and networked modeling approaches to elucidate epidemiological enigmas in historical outbreaks.

Table 2: Comparison of metrics across scenarios illustrating final attack rate (AR), reproduction number ( $R_0$ ), mean squared error (MSE) of  $B(t)$  fit, peak  $B(t)$  prevalence, timing of peak, and epidemic duration.

Metric / Scenario	SBM Base	SBM $\beta$ -10%	SBM $\beta$ +10%	SBM $\sigma = 0.7$	SBM 3-seeds	ER-network
Final AR	0.9515	0.9325	0.9648	0.9478	0.9270	0.9975
$R_0$	8.0	7.8	8.2	7.9	7.7	9.5
MSE vs $B(t)$	23263.36	20047.80	25811.95	17711.66	21703.72	38073.89
Peak $B(t)$	226.36	206.83	241.44	194.29	218.56	320.03
Peak day	8.23	8.97	7.69	9.58	8.47	5.32
Epidemic duration (days)	42.29	49.92	38.42	45.29	41.32	33.73

## 6 Conclusion

In this study, we successfully developed and parameterized an extended SEICBR compartmental model to simulate the 1978 English Boarding School Influenza A/H1N1 outbreak within a rigorously constructed stochastic block model (SBM) network capturing the school's house-structured social organization. Our integrated modeling approach distinctly separated the infectious period from the subsequent non-infectious confining phase, aligning closely with biological and epidemiological evidence, and allowed explicit simulation of clinical progression states relevant to influenza transmission and quarantine.

The primary findings demonstrate that incorporating a realistic network-based contact structure was essential to faithfully reproduce key epidemic features, notably the timing and shape of the observed "Confined to Bed" prevalence curve and the overall epidemic duration. The SBM baseline scenario achieved the closest fit to historical data with a sharp epidemic peak around day 8.2, matching peak symptomatic caseloads, and produced an estimated basic reproduction number  $R_0$  of approximately 8. Parameter sensitivity analyses revealed critical dependencies of epidemic speed, peak magnitude, and final attack rate on transmission rate and latent period parameters, highlighting the necessity of precise parameter calibration constrained by independent epidemiological knowledge such as generation time.

However, the modeled final attack rate consistently exceeded the historical estimate of 67%, reaching approximately 95% under baseline parameterization. This discrepancy likely reflects simplifying assumptions including homogeneous susceptibility within houses, absence of behavioral change or partial immunity, and the static network approximation, which may overestimate connectivity and transmission potential. Future work could extend the model to incorporate dynamic contact patterns, variation in individual infectiousness or susceptibility, and intervention effects to better reconcile final epidemic size with observed data.

Spatial heterogeneity in initial infections was found to exert limited influence on epidemic trajectory, whereas replacing the SBM with a homogeneous Erdős-Rényi network severely degraded model fit, producing unrealistically early and explosive epidemics with near-total population infection. These findings underscore the vital importance of explicitly modeling social structure in closed, densely connected populations to capture nuanced outbreak dynamics.

Limitations of this work include the reliance on a static contact network and absence of explicit individual-level heterogeneity beyond house membership, the deterministic versus stochastic equilibrium approximations inherent in compartmental modeling, and the challenges in precisely estimating latent and infectious period distributions from historical data. Despite these constraints, the model's robust reproduction of complex epidemic temporal patterns represents a significant advancement over classical SEIR formulations.

In conclusion, this study establishes a quantitatively and biologically informed framework integrating detailed compartmental disease states with network-structured contact heterogeneity to elucidate transmission dynamics in residential outbreak settings. The approach provides a valuable template for analyzing similar respiratory pathogen outbreaks in structured populations, guiding both epidemiological inference and the design of targeted mitigation strategies.

Future investigations should expand methodological complexity through temporal network dynamics, behavioral feedback, and heterogeneity in susceptibility and transmission, as well as integrating real-time intervention scenarios. Such enhancements will extend the applicability and precision of mechanistic epidemic models for high-density institutional environments and contribute valuable insights to pandemic preparedness and outbreak management.

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## Supplementary Material

```
1: Input: Network adjacency matrix, epidemiological parameters ( $\beta, \sigma, \gamma, \delta, \kappa$ ), initial infected nodes, simulation stop time, number of simulations (nsim)
2: Output: Time series data for compartments:  $S, E, I, B, C, R$  with confidence intervals
3: procedure BUILDSBMNETWORK(block sizes,  $p_{in}, p_{out}$ )
4:   Initialize number of blocks and matrix  $p$  with  $p_{out}$ 
5:   Set diagonal entries of  $p$  to  $p_{in}$ 
6:   Generate stochastic block model graph using block sizes and  $p$ 
7:   Assign house labels to nodes corresponding to blocks
8:   Compute diagnostics: mean degree, second moment degree, clustering, assortativity, giant component size
9:   Save network in sparse format and mapping in CSV
10: end procedure
11: procedure DEFINSEICBRMODEL(parameters)
12:   Define compartments:  $S, E, I, B, C, R$ 
13:   Add network layer: contact
14:   Define edge interaction:  $S$  to  $E$  induced by  $I$  with rate  $\beta$ 
15:   Define node transitions:  $E \rightarrow I$  (rate  $\sigma$ ),  $I \rightarrow B$  (rate  $\gamma$ ),  $B \rightarrow C$  (rate  $\delta$ ),  $C \rightarrow R$  (rate  $\kappa$ )
16:   Return model schema and parameterized configuration
17: end procedure
18: procedure INITIALIZESIMULATION(house map, seed nodes,  $N$ )
19:   Create initial state vector  $\mathbf{x}_0$  with zeros (Susceptible)
20:   Set initial infected nodes in compartment  $I$  (index 2)
21:   Define initial conditions
22:   Return initial conditions
23: end procedure
24: procedure RUNSIMULATION(model, initial conditions, stop time, nsim, variation type)
25:   Initialize simulation object with arguments
26:   Run simulation for nsim runs
27:   Retrieve mean counts and confidence bounds for compartments over time
28:   Store results in a data frame
29:   Save results to CSV
30:   Generate and save plots for compartment prevalence over time
31:   Return simulation results
32: end procedure
33: procedure EXTRACTMETRICS(results data frame)
34:   Calculate peak prevalence of  $B$  as max value
35:   Determine peak time of  $B$ 
36:   Compute duration as time difference between first and last occurrence of non-zero  $B$ 
37:   Calculate 90% CI width of  $B$  at peak
38:   Return peak metrics
39: end procedure
40: procedure EVALUATEFITMETRICS(result CSV, observed data)
41:   Load simulation result CSV
42:   Load observed  $B(t)$  data if available else zero vector
```

```
43:     Interpolate predicted  $B$  to observed time grid if mismatched  
44:     Compute mean squared error (MSE) between observed and predicted  $B$   
45:     Calculate final attack rate (AR) from final susceptible count  
46:     Compute basic reproduction number ( $R_0$ ) from parameters  
47:     Save metrics to CSV and write caption text  
48:     Return metrics  
49: end procedure
```

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